**PUBLIC NOTICE**
**Initiation of Risk Assessments for Chemicals in Drinking Water**
**[09/20/07]**

A. Requirements

The Calderon-Sher California Safe Drinking Water Act of 1996 requires the Office of Environmental Health Hazard Assessment (OEHHA) to post notices on its Web site of water contaminants for which it is initiating development of public health goals (PHGs) for the chemicals in drinking water. The law also describes the intent and general context of the PHGs. PHGs are concentrations of chemicals in drinking water that are not anticipated to produce adverse health effects following long-term exposures. OEHHA is required to consider potential adverse effects on members of subgroups that comprise a meaningful proportion of the population, including but not limited to infants, children, pregnant women, the elderly, and individuals with a history of serious illness. The public health goals are non-regulatory in nature but are to be used as the health basis to update the state's primary drinking water standards (maximum contaminant levels, or MCLs) established by the California Department of Public Health (DPH) for chemicals subject to regulation.

The act requires PHGs to be developed for the approximately 88 chemicals for which state or federal MCLs are provided, and review and update the risk assessments that form the basis for the PHGs as appropriate at least every five years. Other chemicals may be added to the list by legislative or interdepartmental request. Opportunities for public comment and peer review are provided.

B. Implementation

OEHHA has published 80 PHGs as of September 2007. Two MCLs, for gross alpha and gross beta radionuclides, represent screening levels for contaminants rather than specific regulatory standards; for these, OEHHA has provided risk assessments and guidance memoranda. The technical support documents for these chemicals are posted on the OEHHA Web site at www.oehha.ca.gov.

In addition, PHG re-evaluations have been completed for eight chemicals. For two of these chemicals (cadmium and glyphosate), a complete new PHG document was prepared and published. For the six other chemicals (chlordane, 1,2-dichloroethane, 1,3-dichloropropene, inorganic mercury, lindane, and thallium), OEHHA concluded that no new information was available on these chemicals that would require significant changes to the PHG document. Memoranda to this effect are available at http://www.oehha.ca.gov/water/reports/index.html. The re-reviews of several other chemicals that were announced previously are in progress. Draft PHG documents for the remaining chemicals with existing MCLs or requests for development of a PHG are in preparation.

Draft PHG documents on three chemicals (chlorite, PCBs, and TCDD) have been posted on the OEHHA Web site for public comment, along with drafts of PHG updates for copper and 2,4-D. A 45-day public comment period is provided after posting of the initial drafts, along with a public workshop to elicit comments and discussion. After

revision with consideration of comments received, a further 30-day public comment period will follow. The final revision will include responses to major comments.

Evaluation is now being initiated for several other chemicals for which PHGs were developed earlier (see Section D), which are being re-reviewed as part of the ongoing PHG update process. Information relevant to the development of PHGs is requested on each of these chemicals.

C. PHGs to be released for public review:

Draft documents for the following "new" chemicals (those with no existing PHG) are in progress and are planned for release for initial public review and comment when they are completed:

- Bromate
- Haloacetic acids
- Hexavalent chromium
- Molinate
- Selenium
- Styrene
- 1,2,3-Trichloropropane
- Trihalomethanes

Toxicity reviews are in progress for the following additional chemicals, for which initiation of review was previously announced:

- Alachlor
- Atrazine and simazine
- Chlorite
- Copper
- 2,4-D (Dichlorophenoxyacetic acid)
- Fluoride
- Lead
- Nitrate/nitrite
- PCBs (Polychlorinated biphenyls)
- Trichloroethylene

D. Initiation of risk assessments

Risk assessment is being initiated for the following list of chemicals:

- Antimony
- Bentazon
- Benzo(a)pyrene
- Cyanide
- Dalapon
- Dibromochloropropane (DBCP)
- 1,2-Dichlorobenzene

- 1,4-Dichlorobenzene
- 1,1-Dichloroethylene
- 1,2-Dichloropropane
- Dinoseb
- Endothall
- Endrin
- Hexachlorocyclopentadiene
- Methoxychlor
- Oxamyl
- Pentachlorophenol
- Picloram
- 1,2,4-Trichlorobenzene
- Trichlorofluoromethane (Freon 11)
- Trichlorotrifluoroethane (Freon 113)

These risk assessments are updates of assessments prepared in the first two years of our program (1997 and 1999). The chemicals had earlier been prioritized for review on the basis of availability of new data and significance as drinking water contaminants, so these chemicals on the final list were considered of lowest priority. Little new data are available for most of them. A brief description of the chemicals is provided below. This announcement solicits the submission of other pertinent information on the contaminants that could assist our office in preparing or updating the risk assessment and deriving a revised PHG.

Information submitted to OEHHA in response to this request should not be proprietary in nature, because all information submitted is a matter of public record. Information should be submitted by November 9, 2007 to:

Thomas Parker
PHG Project
Pesticide and Environmental Toxicology Branch
Office of Environmental Health Hazard Assessment
P.O. Box 4010
Sacramento, California 95812-4010

All data submitted will be considered in the development of the PHG for these chemicals. If substantive revisions to the original PHG documents are required, the draft documents will be available for discussion in a public workshop and public comment will be solicited as described above in Section B. The final risk assessments will be utilized by the DPH for potential revisions to the MCLs for the chemicals in drinking water, as described in more detail on their Web site at http://www.dhs.ca.gov/ps/ddwem/chemicals/chemindex.htm.

E. Descriptions of chemicals or substances for initiation of review:

**Antimony**

In 1997, OEHHA developed a PHG for antimony and its compounds of 20 parts per billion (ppb) in drinking water. Antimony is an element found throughout the earth, but present in small quantities in ores. Since antiquity, antimony has been used for many purposes: as a cosmetic, drug, in utensils and currently as a fire retardant and a component in plastics. Antimony is usually available as a compound, and various antimony compounds behave differently in the environment and also have different effects upon humans. Antimony has been reported as exceeding the MCL in California public drinking water supplies eleven times between June 2002 and June 2006.

Ingestion of one antimony compound will produce severe stomach upset resulting in vomiting. Antimony fumes and dusts inhaled by workers are associated with the development of benign tumors of the lungs, dermatitis and, less commonly, effects on the heart and kidneys. Laboratory animals exposed to antimony by inhalation or ingestion exhibit effects similar to those noted in humans. However, antimony and its compounds are not considered to be carcinogenic. The original PHG for antimony was calculated based on minor clinical signs and a slight decrease in longevity noted in a chronic oral study conducted in rats. Other information used to develop the PHG included estimates of human exposure to antimony derived from measurements taken of levels of antimony in air, food and water. The studies presented below represent a selection of the available data.

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**Bentazon**

A PHG of 200 ppb was developed in 1999 for the herbicide bentazon based on a chronic exposure study in dogs. Bentazon is a benzothiadiazinone contact herbicide that acts as an inhibitor of photosynthetic electron transfer in plants. It has a low binding affinity to soil and thus has been found in ground and surface water in California. The detected levels ranged from 0.01 to 20.0 µg/L in 64 out of the 200 wells sampled. The herbicide degrades quickly in plants and soil (3-21 day half life); therefore any contamination of ground or surface water is likely to result from improper agricultural practices.

In the chronic dog study (Allen *et al.*, 1989), a NOAEL was established at 100 ppm in the food (approximately 3.2 mg/kg-day), and the LOAEL was 400 ppm (approximately 13.1 mg/kg-day). Adverse effects reported were emaciation, loose and/or bloody stools, pale mucous membranes, hematological changes suggestive of anemia, intestinal inflammation, and congestion of the small intestine and spleen. The anemia appeared to be due to blood loss from the gastrointestinal tract. This NOAEL was used to calculate the PHG.

Bentazon was classified by U.S. EPA as "Group E," meaning there was no evidence of carcinogenicity. However, two studies warrant a reexamination of the toxicity of bentazon: one, initiated in response to a perceived cancer cluster in a bentazon manufacturing plant, showed a small but statistically significant increase in prostate and stomach cancers, while the second reported mutagenicity in a fruit fly wing spot test (Ott *et al*., 2006; Kaya *et al.*, 2004). Information on other applicable and relevant studies is requested.

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**Benzo(a)pyrene**

The PHG of 4 parts per trillion (ppt; 0.004 μg/L) for benzo(a)pyrene (B(a)P) was published in December, 1997. B(a)P is one of many polycyclic aromatic hydrocarbons that form when organic matter burns incompletely. As such, B(a)P is a ubiquitous environmental contaminant that can be detected in air, water and soil and food. Exposure may occur due to ingestion, inhalation or dermal contact with contaminated air, water, soil or food.

Adverse effects on the hematological, gastrointestinal and immunological systems and effects on reproduction and development have been attributed to exposure to benzo(a)pyrene. Exposure of experimental animals to B(a)P has resulted in statistically significant increases in skin, lung, and forestomach tumors. A cancer potency factor based on an increase in forestomach tumors in mice was employed to develop the PHG for benzo(a)pyrene.

There are many new genotoxicity and mechanism studies of B(a)P, because B(a)P is considered a prototypical genotoxic carcinogen. Also, since the publication of the PHG for B(a)P, two animal studies have been conducted that provide cancer dose-response data for lifetime oral exposures to B(a)P.

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## Cyanide

The Public Health Goal (PHG) of 150 ppb for cyanide was published by OEHHA in December 1997. Cyanide is a naturally occurring chemical that is found in many commonly consumed foods such as almonds, lima beans, cassava, and mustard. Cyanide may also be present at low concentrations in source water as well as finished municipal water supplies. It can be derived from various industrial operations or produced by burning nitrogen-containing materials. High-doses of cyanide inhibit cellular enzymes resulting in hypoxia. Central nervous system (CNS) effects of prolonged or high-level exposure can include demyelinating lesions of the brain and Parkinsonian-like symptoms. The heart is also sensitive to cyanide induced hypoxia. However, cyanide intoxication from the consumption of drinking water is extremely uncommon.

In the survey of the literature, many additional studies relating to the effects of cyanide have been found since the publication of the PHG in 1997. The new toxicity information appears likely to have significant impact on the existing toxicology and risk assessment sections of the PHG document. The studies presented below represent a selection of the available data.

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**Dibromochloropropane**

A PHG for 1,2-dibromo-3-chloropropane (DBCP) of 1.7 ppt was developed by OEHHA in 1999, based on carcinogenicity in experimental animals in multiple toxicity studies. DBCP was used as a soil fumigant for multiple crops until suspension of its use in 1977 and final cancellation in 1985. DBCP migrated readily to groundwater and is very persistent in the aquifer; it has contaminated groundwater over a wide area in California's Central Valley. Although the most heavily contaminated sources are not used for drinking water, they may still be used for irrigation. Because of the volatility of DBCP, significant residues in crops are not expected, but some evaporation to air will occur.

DBCP caused forestomach cancers in rats and mice after gavage administration, as well as mammary gland tumors in female rats. It also caused oral and nasal cavity tumors after inhalation exposures in rats and mice, and lung tumors in mice. It is mutagenic and clastogenic. DBCP inhibits sperm production in humans and animals, and has been associated with human infertility after occupational exposures. The few studies of DBCP effects since publication of the PHG in 1999 may provide significant new perspective on mechanism of action of DBCP.

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**1,2-Dichlorobenzene**

A PHG of 0.6 mg/L (600 ppb) was developed in 1997 for 1,2-dichlorobenzene (1,2-DCB, also known as ortho-dichlorobenzene) in drinking water. The PHG is based on several adverse effects, including hepatotoxicity and organ and body weight changes observed in experimental animals in a subchronic oral exposure study. 1,2-DCB is a solvent and a chemical intermediate. It also has been used as an insecticide/fumigant; the last

registered pesticide product containing 1,2-DCB as an active ingredient was removed from the California market in 1985.

1,2-DCB and other simple chlorinated benzenes have been used in the study of hepatocyte injury and repair mechanisms, which may provide some perspective on both the dose-response and structure-activity relationships for these chemicals (see also 1,4-DCB). Of particular interest is why 1,2-DCB causes hepatotoxicity but has not been found to cause liver tumors, while 1,4-DCB causes both hepatotoxicity and a clearly increased incidence of liver tumors.

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**1,4-Dichlorobenzene**

A PHG of 0.006 mg/L (6 ppb) was developed in 1997 for 1,4-dichlorobenzene (1,4-DCB), also known as para-dichlorobenzene, in drinking water. The PHG is based on hepatocarcinogenic effects in both male and female mice observed in a chronic study conducted by the National Toxicology Program (NTP). 1,4-DCB is used mainly as a fumigant for the control of moths, molds, and mildews (mothballs), and as a space deodorant for toilets and refuse containers. It is also used as an intermediate in the production of other chemicals, in the control of tree-boring insects, and in the control of mold in tobacco seeds.

The general population is exposed to 1,4-DCB through breathing vapors from household products such as mothballs and toilet deodorizer blocks. Traces of 1,4 DCB have been found in foods such as meat, eggs, and honey. Acute human exposure to 1,4-dichlorobenzene in air results in irritation to the eyes, skin, and throat. Subchronic exposure to experimental animals caused liver effects (degeneration and necrosis), bone

marrow hypoplasia, and renal damage and necrosis at all levels of ingestion. Chronic exposure to 1,4 DCB in mice showed renal damage, liver toxicity, increases in heart, liver, and lung weights, and parathyroid hyperplasia among males and neuropathy in females.

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### 1,1-Dichloroethylene

A PHG of 0.01 mg/L (10 μg/L, or 10 ppb) was developed in 1999 for 1,1-dichloroethylene (1,1-DCE, also known as vinylidene chloride) in drinking water. 1,1-DCE is used principally for the production of polyvinylidene chloride polymers. 1,1-DCE does not occur naturally, but can be found in landfills as a result of the breakdown of polyvinylidene chloride products. Owing to its high vapor pressure, air releases are the largest sources of the chemical in the environment. Concentrations of 1 to 550 μg/mL 1,1-DCE have been reported in surface waters near industrial sites.

The 1999 PHG was based on the most sensitive toxic endpoint, midzonal hepatocellular fatty changes in female rats at 50 ppm (approximately 9 mg/kg-day) and greater in their drinking water. There appears to be inadequate evidence to consider 1,1-DCE to be a carcinogen. Inhalation exposure to DCE is both fetotoxic and produces developmental effects in laboratory animal studies at maternally toxicity doses. Other recent studies on lung and immune system effects of 1,1-DCE deserve additional consideration.

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### 1,2-Dichloropropane

A PHG of 0.5 µg/L (or ppb) was developed in 1999 for 1,2-dichloropropane (1,2-DCP) in drinking water, based on carcinogenic effects observed in experimental animals. 1,2-DCP, also known as propylene dichloride, is primarily used as a chemical intermediate in the synthesis of other chlorinated hydrocarbons. In the past, it was used as a fumigant and industrial solvent. It may be found as a contaminant in the fumigant Telone (1,3-dichloropropene).

Kidney and liver damage has been observed after acute and subchronic exposure to 1,2-DCP, both in humans and in experimental animals. Tests for mutagenicity and genotoxicity have been mixed, some negative, some positive. 1,2-DCP has been frequently detected at low levels in air as well as ground and surface water, although levels should have decreased due to improved industrial vapor controls and discontinuation of its use as a fumigant. The recent research on reproductive system effects of 1,2-DCP in female rats particularly deserves review.

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**Dinoseb**

A PHG of 14 ppb was developed for dinoseb (2-sec-butyl-4,6-dinitrophenol) in drinking water in 1997. Dinoseb is a dinitrophenolic compound once used extensively as an herbicide and pesticide in California. It was suspended for all pesticide uses by U.S. EPA in 1986 based on concern about potential reproductive and teratogenic effects in agricultural workers. It has not been detected in California water supplies in several years.

Dinoseb, like other dinitrophenols, is an inhibitor of mitochondrial respiration (the source of most cellular energy, used to support metabolism). More recent studies of this effect have provided new perspectives on its spermatocidal and teratogenic effects.

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## Endothall

A PHG of 580 ppb for endothall (7-oxabicyclo(2.2.1) heptane-2,3 dicarboxylic acid) in drinking water was developed in 1997 based on gastric toxicity observed in dogs. Endothall is an organic acid effective as a contact weed killer. Major uses include the defoliation of cotton, control of algae and aquatic weeds and as a desiccant on other crops. Its selective action and rapid breakdown to nontoxic products makes it desirable for control of aquatic weeds, leaving other desirable species such as fish and insects relatively unaffected.

As a strong organic acid, endothall is poorly absorbed both orally and dermally, and its effects tend to be limited to direct irritancy. However, endothall is considered to be a selective inhibitor of type 2A protein phosphatase (PP2A(c)), and has been used for this purpose in metabolic studies. The recent reevaluation of endothall for reregistration by U.S. EPA (2005) included extra consideration of exposures for infants and children, and concluded that drinking water exposure for infants less than one year old is at the level of concern (using the U.S. EPA MCL of 100 ppb as a surrogate for actual drinking water exposure data). OEHHA will evaluate this conclusion in the context of our mandate for protecting sensitive populations.

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**Endrin**

A PHG of 0.0018 mg/L (1.8 ppb) was developed for endrin in drinking water in 1999. The PHG is based on the observation of seizures and minor pathological changes to the liver in dogs fed 2 and 4 ppm endrin for two years. Endrin is an environmentally persistent chlorinated hydrocarbon pesticide similar to aldrin and dieldrin, which are classified as carcinogens. It has been postulated that the failure to demonstrate carcinogenicity of the structurally related compound endrin is that the high toxicity of endrin limits the daily dose to levels at which the tumor incidence would be insignificant. The biological half-life of endrin in mammals is also quite short, compared to chlorinated hydrocarbons like dieldrin and DDT. Mechanistic data that might help resolve the question of its potential carcinogenicity has not been produced since the publication of the earlier PHG.

Most uses of endrin were cancelled by the U.S. EPA in 1979, and the remaining limited usage was cancelled in 1991, so there has been little incentive for further toxicity studies. Endrin residues persist in the environment in aqueous sediments, soil, and marine fish, but not in red meat and poultry.

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### Hexachlorocyclopentadiene

A PHG of 0.05 mg/L (50 ppb) was developed in 1999 for hexachlorocyclopentadiene (HCCPD) in drinking water, based on stomach lesions after oral administration of HCCPD to rats and mice. HCCPD is a halogenated hydrocarbon used as an intermediate in the production of dyes, resins, pharmaceuticals, flame retardants, and insecticides. HCCPD is also used in the production of a variety of industrial chemicals, including ketones, fluorocarbons, acids, esters, and plastics. The major toxic effect of HCCPD is irritation at the point of contact (lungs, skin, or stomach). Kidney and liver damage also occurs at moderate doses after oral (gavage) administration to rodents.

Our preliminary literature scan revealed no new toxicity studies on this chemical. The updated U.S. EPA review takes an approach similar to the 1987 U.S. EPA review (and the 1999 PHG), except for a benchmark calculation of the reference dose.

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**Methoxychlor**

In 1999, OEHHA published a PHG of 0.03 mg/L (30 ppb) for methoxychlor (MXC) in drinking water. MXC is a chlorinated hydrocarbon insecticide that is approved for use on livestock as well as on numerous agricultural crops. MXC was suspended from use in California in 1995 and in the rest of the country in 2000; U.S. EPA revoked all tolerances in crops in 2002. MXC has been found in surface waters near points of application for pest control, and in groundwater near waste disposal sites; it has not been detected in California municipal drinking water. Very high doses of MXC can cause tremors, convulsions, and other signs of neurological stimulation. The most significant effects of repeated exposure to MXC are on reproductive tissues; both the parent compound and its metabolites exhibit estrogenic activity. Chronic and sub-chronic exposure can produce adverse effects on the male and female reproductive system, developmental effects, and long-term effects on neurobehavioral development. Carcinogenicity studies on MXC are inadequate; both the U.S. EPA and IARC have judged MXC to be not classifiable as to human carcinogenicity. The PHG for MXC is based on reproductive effects in female rats exposed during the perinatal period. The data are supported by similar findings in other reproductive and developmental studies in animals. No human studies on human sensitivity to the potential endocrine-disruptive effects of this pesticide are available.

A large volume of new literature, predominantly relating to the estrogenic effects of MXC and its metabolites in animal studies, has been published since the PHG was developed in 1999. Several new mechanism studies are also available. Reported reproductive tract abnormalities at doses lower than those used to derive the original PHG also deserve intensive review. Representative new studies available are listed below.

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**Oxamyl**

A PHG of 50 ppb was developed for oxamyl (S-methyl N'N'-dimethyl-N-[(methylcarbamoyl) oxy]-1-thiooxamimidate) in drinking water in 1997. Oxamyl is a carbamate insecticide which acts by inhibition of the enzyme acetylcholinesterase. Its

acute toxicity is very high. The risk assessment is based on decreased body weight gain, presumably caused by the ill effects of cholinesterase inhibition. Oxamyl is readily degraded in soil and groundwater; it has rarely been detected in California water sources, although it has been reported at low levels in drinking water from several other states.

The inhibition of cholinesterase caused by carbamates is very short-acting, so acute effects are most relevant. New studies by Malley *et al.* (1997, 1998) involving gavage administration of oxamyl to rats appear to provide a much lower no-observed-adverse-effect level than previous studies, and are likely to lead to a decreased PHG. The calculation should be based on exposure of an infant or toddler, because these age groups consume much greater amounts of water per body weight equivalent than do adults, and thus represent a susceptible population.

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## Pentachlorophenol

The PHG for pentachlorophenol (PCP) of 0.4 parts per billion was developed in 1997, and is based on carcinogenic effects in mice. PCP has primarily been used as a pesticide, for wood treatment and as a disinfectant. Wood used for decks, railings, and playground structures was frequently treated with PCP. Widespread human exposure occurred from dermal and inhalation exposures, as well as hand-to-mouth contact by children. PCP also leached into surface and groundwater. Because of its hazards, PCP is now restricted to use in a heat and pressure wood treatment process, for special outdoor applications such as utility poles.

In animal studies, short-term to chronic treatment with PCP has been reported to cause adverse effects on kidney and liver, increased fetal reabsorptions, anemia and leukopenia, and thyroid hormone disruption at relatively low levels (NOAELs <10 mg/kg-day). Significant increases in tumors in mice occur at about 20 mg/kg-day or more. A relatively large number of toxicity studies have been published since the release of the original PHG document in 1997. Major toxicity reviews have been published by ATSDR and U.S. EPA. OEHHA has also reviewed PCP for development of a child-specific reference dose for school siting and a No Significant Risk Level for Proposition 65.

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**Picloram**

A PHG of 500 ppb was developed for picloram (4-amino-3,5,6-trichloropicolinic acid) in drinking water in 1997. Picloram is a polychlorinated herbicide that has been widely used for the control of broad-leaved weeds and woody plants along rights-of-way. It is applied alone or in combination with 2,4-dichlorophenoxyacetic acid (2,4-D) in a product named Tordon.

Picloram is chemically related to the herbicides clopyralid and triclopyr; these chemicals are classified as auxins, which are plant growth regulators. Like other herbicides such as 2,4-D, these chemicals disrupt plant growth regulation, leading to overgrowth and death of susceptible plants. Mammalian toxicity is low. The PHG was based on increased liver weight in a 6-month dog-feeding study with a no-observed-adverse-effect level of 7 mg/kg-day and a lowest-observed-effect level of 35 mg/kg-day. Cancer studies have been negative except in some cases for liver tumors that were attributed to contamination of the administered product with hexachlorobenzene.

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### 1,2,4-Trichlorobenzene

A PHG of 0.005 mg/L (0.005 ppm, or 5 ppb) was developed in 1999 for 1,2,4-trichlorobenzene (1,2,4-TCB) in drinking water, based on enlargement of adrenal glands in a subchronic rat study. 1,2,4-TCB is used as a solvent in chemical manufacturing, a dye carrier for textiles, an intermediate in the production of other chemicals, a degreasing agent, a component of dielectric fluids, and as a component of lubricants and oils.

OEHHA recently reviewed 1,2,4-TCP for potential listing as a carcinogen under Proposition 65. A chronic study showed that male and female mice administered 1,2,4-TCB in their diet developed a high incidence of hepatocellular adenomas and carcinomas. A similar study in rats showed no evidence of carcinogenic effects, and a skin painting study in mice produced no evidence of carcinogenicity. Genotoxicity tests have mostly been negative, and dose-related increases in micronuclei have been negative. Considering this evidence, the expert committee concluded that 1,2,4-TCP should not be listed as a human carcinogen.

The 1999 PHG included a 10-fold factor for potential carcinogenicity of 1,2,4-TCB, based on preliminary reports of the 1994 CMA studies, which resulted in a total uncertainty factor (UF) of 10,000. Our present practice is to limit the total UF to 3,000. OEHHA acknowledges the potential concern about carcinogenicity from exposure to this halogenated benzene compound, and solicits further input on the approach for the planned PHG update.

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**Trichlorofluoromethane (Freon 11)**

A PHG of 0.7 mg/L (700 ppb) for trichlorofluoromethane (Freon 11) was developed in 1997. Freon 11 was one of the most widely used chlorofluorocarbons in industrial applications, including use as a blowing agent in foam production and as an aerosol propellant. Because of its effects on the atmospheric ozone layer, U.S. production was banned in 1996. A California PHG and MCL were developed because of its leaching from waste sites, and potential for water supply contamination.

The most well-known toxic effects of Freon 11 include cardiac and pulmonary disturbances (e.g., cardiac arrhythmias, tachycardia and hypotension) and changes in respiratory parameters, which were elucidated because of inhalation abuse of aerosol products. Other effects include hepatic lesions, central nervous system dysfunction, and skin and eye irritation. Chronic animal exposure studies were negative for carcinogenicity. Limited information is available on other potential effects, and our preliminary literature survey revealed no new toxicity studies.

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**Trichlorotrifluoroethane (Freon 113)**

A Public Health Goal (PHG) of 4 mg/L (4 ppm) was developed for 1,1,2-trichloro-1,2,2-trifluoroethane (Freon 113) in drinking water in 1997. Freon 113 was widely used in industrial applications as a solvent for degreasing and dry cleaning, as a refrigerant, in

fire extinguishers, as a chemical intermediate and as a blowing agent in foam production. Because of its effects on the atmospheric ozone layer, U.S. production was banned in 1996. Exposures continued due to its persistence in air and presence in waste-water streams and leachate from waste sites, leading to the development of the California PHG and MCL.

Freon 113 has extremely low acute toxicity, but cardiac sensitization was reported from inhalation abuse of aerosol products, and confirmed in dog studies. Repeated short-term administration in several species did not reveal any distinctive toxicological effects. A rat chronic inhalation study resulted in increases in liver weight, but no carcinogenicity.

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